WEST

Help | Logout | Interrupt

Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences | Cases

Search Results -

Terms	Documents
L5 and (antisense or ribozyme$)	10

Database: US Patents Full-Text Database
US Pre-Grant Publication Full-Text Database
JPO Abstracts Database
EPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L6

Refine Search

Recall Text | Clear

Search History

DATE: Friday, June 13, 2003 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; PLUR=NO; OP=OR			
L6	L5 and (antisense or ribozyme$)	10	L6
L5	L4 and reject$	39	L5
L4	L3 and (vcam or elam or icam)	39	L4
L3	cornea$ same allograft$	326	L3
L2	L1 and (icam or elam or vcam)	10	L2
L1	cornea$ same explant$	103	L1

END OF SEARCH HISTORY

WEST

Help | Logout | Interrupt

Main Menu | Search Form | Posting Counts | Show S Numbers | Edit S Numbers | Preferences | Cases

Search Results -

Terms	Documents
L4 and reject$	39

Database: US Patents Full-Text Database; US Pre-Grant Publication Full-Text Database; JPO Abstracts Database; EPO Abstracts Database; Derwent World Patents Index; IBM Technical Disclosure Bulletins

Search: L5

Refine Search | Recall Text | Clear

Search History

DATE: Friday, June 13, 2003 Printable Copy Create Case

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DB=USPT,PGPB,JPAB,EPAB,DWPI,TDBD; PLUR=NO; OP=OR			
L5	L4 and reject$	39	L5
L4	L3 and (vcam or elam or icam)	39	L4
L3	cornea$ same allograft$	326	L3
L2	L1 and (icam or elam or vcam)	10	L2
L1	cornea$ same explant$	103	L1

END OF SEARCH HISTORY

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(c) 2003 American Chemical Society
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          130403  EXPLANT?
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?s s1 (s) (icam or elam or vcam)
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           71655  ICAM
            4900  ELAM
           24078  VCAM
      S2      10  S1 (S) (ICAM OR ELAM OR VCAM)
?rd
...completed examining records
      S3       4  RD (unique items)
?show files;ds;t/3,k/all
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Set	Items	Description
S1	1177	CORNEA? (S) EXPLANT?
S2	10	S1 (S) (ICAM OR ELAM OR VCAM)
S3	4	RD (unique items)

>>>KWIC option is not available in file(s): 399

3/3,K/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

14163665 BIOSIS NO.: 200300157694
Intercellular adhesion molecule-1 expression on human corneal epithelial outgrowth from limbal explant in culture.
AUTHOR: Iwata M(a); Fushimi N; Suzuki Y; Suzuki M; Sakimoto T; Sawa M
AUTHOR ADDRESS: (a)Department of Ophthalmology, Nihon University, School of Medicine, 30-1 Ohyaguchikami-machi, Itabashi-ku, Tokyo, 173-8610, Japan** Japan
JOURNAL: British Journal of Ophthalmology 87 (2):p203-207 February 2003 2003
MEDIUM: print
ISSN: 0007-1161
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Aim: To investigate the relation between intercellular adhesion molecule (*ICAM*)-1 expression and cellular dynamics occurring concomitantly with epithelial cell movement. Methods: Outgrowing epithelial sheets of human *corneal* epithelial (HCE) cells from cultured limbal *explants* were examined by immunoperoxidase staining with anti-*ICAM*-1 monoclonal antibody. An adhesion assay was performed using the epithelial sheets of HCE cells and an Epstein-Barr virus (EVB) infected B cell lymphoma cell line (EVB+BJAB) expressing CD11a/CD18, a counter-receptor of *ICAM*-1. Also, the effect of calphostin C, a specific protein kinase C (PKC) inhibitor, on *ICAM*-1 expression on the outgrowing epithelial sheets of HCE cells was examined. Results: Strong positive staining for *ICAM*-1 was found predominantly on HCE cells in the marginal segment of the epithelial sheet, particularly on the cells at the leading edge. EBV+BJAB cells adhering to the HCE cells corresponded well to the area of *ICAM*-1 staining. Treatment of outgrowing epithelial sheets with calphostin C markedly decreased the *ICAM*-1 expression on the HCE cells. Conclusion: *ICAM*-1 is actively expressed on HCE cells in the marginal segment of the outgrowing epithelial sheets where there is active movement mediated through a PKC dependent mechanism, suggesting the role of *ICAM*-1 in epithelial cell motility such as the spreading and migration of cells.

3/3,K/2 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

07614223 Genuine Article#: 178MF No. References: 0
Title: Dexamethasone inhibs cytokine induced *ICAM* and VEGF expression in human *corneal* *explants*
Author(s): Sims MG; Tsai SY; Lai CI; Bowen TA; Bouchard CS
Corporate Source: LOYOLA UNIV,MED CTR, PROGRAM NEUROSCI, DEPT CELL BIOL NEUROBIOL & ANAT/NEW ORLEANS//LA/70118; LOYOLA UNIV,MED CTR, DEPT OPHTHALMOL/NEW ORLEANS//LA/70118; CHICAGO COLL OSTEOPATH MED,/CHICAGO//IL/
Journal: INVESTIGATIVE OPHTHALMOLOGY & VISUAL SCIENCE, 1999, V40, N4 (MAR 15), P36244-36244
ISSN: 0146-0404 Publication date: 19990315
Publisher: ASSOC RESEARCH VISION OPHTHALMOLOGY INC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998
Language: English Document Type: MEETING ABSTRACT

Title: Dexamethasone inhibs cytokine induced *ICAM* and VEGF expression in human *corneal* *explants*

3/3,K/3 (Item 1 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)
(c) 2003 American Chemical Society. All rts. reserv.

138332860 CA: 138(22)332860v PATENT
Antisense oligonucleotide targeting cell adhesion molecules for preserving corneal explants and preventing corneal allograft rejection
INVENTOR(AUTHOR): Bennett, C Frank; Mirabelli, Christopher K.
LOCATION: USA
ASSIGNEE: Isis Pharmaceuticals, Inc.
PATENT: PCT International ; WO 200332920 A2 DATE 20030424
APPLICATION: WO 2002US33236 (20021016) *US 982262 (20011018)
PAGES: 106 pp. CODEN PIXXD2 LANGUAGE English CLASS: A61K-000/A
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; OM; PH; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TN; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZM; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ; UG; ZM; ZW; AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG

3/3,K/4 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2003 American Chemical Society. All rts. reserv.

138331722 CA: 138(22)331722q PATENT
Oligonucleotide modulation of cell adhesion for preserving corneal explants and preventing corneal allograft rejection
INVENTOR(AUTHOR): Bennett, C. Frank; Mirabelli, Christopher
LOCATION: USA
PATENT: U.S. Pat. Appl. Publ. ; US 20030077565 A1 DATE: 20030424
APPLICATION: US 982262 (20011018) *US 7997 (19930121) *US 969151 (19930210) *US 63167 (19930517) *US 440740 (19950512) *US 128496 (19980803) *US 659288 (20000912)
PAGES: 58 pp., Cont.-in-part of U.S. Ser. No. 659,288, abandoned.
CODEN: USXXCO LANGUAGE: English CLASS: 435002000; A61K-048/00A; A01N-001/02B

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          4900  ELAM
         24078  VCAM
         71655  ICAM
        154052  ANTISENSE
         31644  RIBOZYME?
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                RIBOZYME?)
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...completed examining records
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S2	10	S1 (S) (ICAM OR ELAM OR VCAM)
S3	4	RD (unique items)
S4	2	S1 AND (ELAM OR VCAM OR ICAM) AND (ANTISENSE OR RIBOZYME?)
S5	2	RD (unique items)

>>>KWIC option is not available in file(s): 399

5/3,K/1 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
(c) 2003 American Chemical Society. All rts. reserv.

138332860 CA: 138(22)332860v PATENT
Antisense oligonucleotide targeting cell adhesion molecules for preserving corneal explants and preventing corneal allograft rejection
INVENTOR(AUTHOR): Bennett, C. Frank; Mirabelli, Christopher K.
LOCATION: USA
ASSIGNEE: Isis Pharmaceuticals, Inc.
PATENT: PCT International ; WO 200332920 A2 DATE: 20030424
APPLICATION: WO 2002US33236 (20021016) *US 982262 (20011018)
PAGES: 106 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-000/A
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; OM; PH; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TN; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZM; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW ; MZ; SD; SL; SZ; TZ; UG; ZM; ZW; AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG

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DIALOG(R)File 399:CA SEARCH(R)
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138331722 CA: 138(22)331722q PATENT
Oligonucleotide modulation of cell adhesion for preserving corneal explants and preventing corneal allograft rejection
INVENTOR(AUTHOR): Bennett, C. Frank; Mirabelli, Christopher
LOCATION: USA
PATENT: U.S. Pat. Appl. Publ. ; US 20030077565 A1 DATE: 20030424
APPLICATION: US 982262 (20011018) *US 7997 (19930121) *US 969151 (19930210) *US 63167 (19930517) *US 440740 (19950512) *US 128496 (19980803) *US 659288 (20000912)
PAGES: 58 pp., Cont.-in-part of U.S. Ser. No. 659,288, abandoned.
CODEN: USXXCO LANGUAGE: English CLASS: 435002000; A61K-048/00A; A01N-001/02B

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          184996  ALLOGRAFT?
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           24078  VCAM
            4900  ELAM
           71655  ICAM
      S7      94  S6 AND (VCAM OR ELAM OR ICAM)
?s s6 and (antisense or ribozyme?)
            3095  S6
          154052  ANTISENSE
           31644  RIBOZYME?
      S8       4  S6 AND (ANTISENSE OR RIBOZYME?)
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Set	Items	Description
S1	1177	CORNEA? (S) EXPLANT?
S2	10	S1 (S) (ICAM OR ELAM OR VCAM)
S3	4	RD (unique items)
S4	2	S1 AND (ELAM OR VCAM OR ICAM) AND (ANTISENSE OR RIBOZYME?)
S5	2	RD (unique items)
S6	3095	CORNEA? (S) ALLOGRAFT?
S7	94	S6 AND (VCAM OR ELAM OR ICAM)
S8	4	S6 AND (ANTISENSE OR RIBOZYME?)
S9	4	RD (unique items)

>>>KWIC option is not available in file(s): 399

9/3,K/1 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
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138332860 CA: 138(22)332860v PATENT
Antisense oligonucleotide targeting cell adhesion molecules for preserving corneal explants and preventing corneal allograft rejection
INVENTOR(AUTHOR): Bennett, C. Frank; Mirabelli, Christopher K.
LOCATION: USA
ASSIGNEE: Isis Pharmaceuticals, Inc.
PATENT: PCT International ; WO 200332920 A2 DATE: 20030424
APPLICATION: WO 2002US33236 (20021016) *US 982262 (20011018)
PAGES: 106 pp. CODEN: PIXXD2 LANGUAGE: English CLASS: A61K-000/A
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; OM; PH; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TN; TR; TT; TZ; UA; UG; US; UZ; VN; YU; ZA; ZM; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW ; MZ; SD; SL; SZ; TZ; UG; ZM; ZW; AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG

9/3,K/2 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
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138331722 CA: 138(22)331722q PATENT
Oligonucleotide modulation of cell adhesion for preserving corneal explants and preventing corneal allograft rejection
INVENTOR(AUTHOR): Bennett, C. Frank; Mirabelli, Christopher
LOCATION: USA
PATENT: U.S. Pat. Appl. Publ. ; US 20030077565 A1 DATE: 20030424
APPLICATION: US 982262 (20011018) *US 7997 (19930121) *US 969151 (19930210) *US 63167 (19930517) *US 440740 (19950512) *US 128496 (19980803) *US 659288 (20000912)
PAGES: 58 pp., Cont.-in-part of U.S. Ser. No. 659,288, abandoned.
CODEN: USXXCO LANGUAGE: English CLASS: 435002000; A61K-048/00A; A01N-001/02B

9/3,K/3 (Item 1 from file: 442)
DIALOG(R)File 442:AMA Journals
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00115965
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Corneal Dystrophies of Epithelial Genesis The Possible Therapeutic Use of Limbal Stem Cell Transplantation (ARTICLE)

Archives of Ophthalmology
JAN, 2001; Editorial: tzh120
LINE COUNT: 00327

... Retroviral transduction of the cultured cells could prevent production of the defective keratoepithelin by targeting the messenger RNA carrying the mutation. Such an approach using *ribozymes* has been used to target mutated rhodopsin in animal models of retinitis pigmentosa.29/ The proof of principle for this approach has been provided in...155-171.

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Tsubota K, Satake Y, Ohyama M, et al. Surgical reconstruction of the ocular surface in advanced ocular cicatricial pemphigoid and...

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Bradshaw JJ, Obritsch WF...

9/3,K/4 (Item 2 from file: 442)
DIALOG(R)File 442:AMA Journals

00115712

Topical Soluble Tumor Necrosis Factor Receptor Type I Suppresses Ocular Chemokine Gene Expression and Rejection of Allogeneic Corneal Transplants (ARTICLE)

QIAN, YING; DEKARIS, IVA; YAMAGAMI, SATORU; DANA, M. REZA
Archives of Ophthalmology
Dec, 2000; Laboratory Sciences: tzo1666
LINE COUNT: 00573

... sTNFR-I- and vehicle only-treated groups was determined using a multiprobe ribonuclease protection assay. Results: Hosts treated with topical sTNFR-I experienced significantly enhanced *corneal* *allograft* survival compared with animals treated with vehicle alone (P=.01). Moreover, postoperative messenger RNA levels of RANTES and macrophage inflammatory protein-1B in sTNFR-I-treated eyes were substantially suppressed compared with vehicle-treated eyes. Vehicle-treated eyes bearing rejected *allografts* expressed higher levels of messenger RNA for both chemokines than control eyes bearing accepted *allografts*. Conclusions: Topical treatment with sTNFR-I promotes the acceptance of allogeneic corneal transplants and inhibits gene expression of 2 chemokines (RANTES and macrophage inflammatory protein-1B) associated with corneal graft

rejection. Clinical Relevance: Our findings support the feasibility of a topical anticytokine strategy as a means of reducing *corneal* *allograft* rejection without resorting to the use of potentially toxic immunosuppressive drugs. Arch Ophthalmol. 2000;118:1666-1671
DESPITE the overall success of *corneal* transplantation, immunologic rejection remains the principal threat to *allograft* longevity.1/ Although not all of the cellular and molecular components that mediate *corneal* graft rejection have been identified to date, there is increasing evidence that the proinflammatory cytokine tumor necrosis factor <unprintable> (TNF-<unprintable>) is involved in the...
... Based on these data, it has been proposed2,8,13/ that TNF-<unprintable> could serve as an appropriate target for therapeutic intervention in prevention of *corneal* *allograft* rejection. It has already been shown that administration of anti-TNF-<unprintable> therapy can be effective in treatment of immune-mediated diseases, including arthritis14-17...

... can be associated with untoward adverse effects, in this study we investigated the effect of topical soluble TNFR-I (sTNFR-I) on the survival of *corneal* *allografts* . Soluble TNFR-I has been shown to profoundly suppress bioactivity of TNF-<unprintable> by binding free TNF-<unprintable> and preventing ligation of the membrane-bound receptors.17/
Corneal *allograft* rejection is pathologically characterized by leukocytic infiltration into the graft stroma and adherence of mononuclear cells to the donor *corneal* endothelium.12/ Recruitment of immune and inflammatory cells to the target tissue, including *allografts*, has been associated with the function of specialized chemotactic cytokines known as chemokines.25/ Studies have shown that *corneal* transplant rejection is associated with profound up-regulation in ocular gene expression of CC (B) chemokines, including macrophage inflammatory protein-1 alpha, macrophage inflammatory protein...

... also interested in determining whether local suppression of TNF-<unprintable> activity by application of sTNFR-I could alter gene expression of the chemokines associated with *corneal* transplant rejection.
RESULTS
ORTHOTOPIC *CORNEAL* *ALLOGRAFT* SURVIVAL
A total of 50 corneas from B10.D2 mice were grafted orthotopically onto 50 BALB/c mice, of which 30 were randomized to receive...

... treated grafted eyes did not reveal any statistically significant differences.
COMMENT
In the present study, prophylactic administration of topical sTNFR-I enhanced the survival of *corneal* *allografts* disparate at multiple minor H antigens. These findings, coupled with the previous observation2/ that hosts with a genetic deficiency of TNFR-I exhibit a profound increase in the survival rate of minor H-mismatched *corneal* grafts, strongly confirm TNF-<unprintable> as an important mediator in the pathogenesis of *corneal* *allograft* rejection. We focused our attention on alloimmunity to minor antigens because it has been shown that disparity at the level of minor antigens provides a significantly more formidable immune barrier to *corneal* graft acceptance than disparity at major histocompatibility complex loci.29/ Accordingly, Sonoda and Streilein30/ showed that grafts disparate at multiple minor H antigens are rejectedon the induction and expression of *corneal* alloimmune mechanisms remains incompletely understood. In the murine orthotopic *corneal* transplantation model, peak secretion of TNF-<unprintable> protein in allogeneic grafts is observed 1 to 2 weeks after surgery,8/ whereas graft rejection typically occurs...

... phase of alloimmunity is supported by previous data13/ from our laboratory showing that the migration of professional antigen-presenting cells (including Langerhans cells) into the *cornea* is largely mediated by TNF-<unprintable>. The critical role of antigen-presenting cell migration in indirect sensitization of T cells to *corneal* transplants31,32/ is emphasized by data33/ showing that suppression of Langerhans cell trafficking into *corneal* grafts can prevent host sensitization to the

transplants. Accordingly, we propose that local neutralization of TNF-<unprintable> activity by application of sTNFR-I imposes its beneficial effect on *allograft* survival, at least in part, by inhibiting leukocyte recruitment in the early postoperative period.

As shown previously,13/ TNF-<unprintable> need not effect its chemoattractant. .

... chemokine mRNA in grafted hosts treated with sTNFR-I and vehicle alone. Consistent with previous findings26/ in major histocompatibility complex and minor H fully mismatched *corneal* transplantation, increased gene expression of RANTES and MIP1B is associated with rejection of minor H-disparate *corneal* grafts. More important, our results clearly show that sTNFR-I therapy applied after *corneal* transplantation significantly down-regulates local gene expression of RANTES and MIP1B. These ligands, by virtue of binding to the widely expressed CCR1 and CCR5 receptors...

... as critical chemoattractants for antigen-presenting cells and activated CD4/+/ T cells.6,7,37,38/ Preliminary data from our laboratory in nontransplant models of *corneal* inflammation show a significant suppression of *corneal* dendritic cell migration in response to *corneal* injury after treatment with sTNFR-I (unpublished observations). Our present data demonstrating that sTNFR-I treatment can suppress expression of RANTES and MIP1B below that for accepted untreated *corneas* suggest that the depressed level of chemokine expression is not simply secondary to suppression of the rejection process but rather a mechanism by which sTNFR-I can mediate its beneficial effect on *allograft* survival. Taken together, these data support our hypothesis that sTNFR-I can down-modulate the induction and expression phases of alloimmunity by suppressing leukocyte recruitment...this represents the first study providing evidence for local anti-TNF strategies, using the novel method of topically administering sTNFR-I, for effective prevention of *corneal* *allograft* rejection. Various forms of recombinant sTNFR-I, including monomeric 4 domain, monomeric 2.6 domain, and 30-kd polyethylene glycol-linked 2.6 domain molecules...

... surface that is also well tolerated because of its biophysical properties and hence can increase the availability of pharmacotherapeutic agents to the ocular surface and *cornea*.41,42/

Currently available preventive and therapeutic regimens for corneal transplant rejection are associated with significant complications.43/ Hence, it is desirable to devise intervention...

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MATERIALS AND METHODS

ANIMALS

Six- to 8-week-old male BALB/c mice (Taconic Farms Inc, Germantown, NY) and... a multiprobe ribonuclease protection assay system (PharMingen, San Diego, Calif) as recommended by the supplier. Briefly, a mixture of [<unprintable>-/32/P]uridine triphosphate-labeled *antisense* riboprobes was generated from the chemokine template set mCK-5 (PharMingen). Fifteen micrograms of total RNA was used in each sample. Total RNA was hybridized overnight at 56 degreesC with 300 pg of the /32/P *antisense* riboprobe mixture. Nuclease-protected RNA fragments were purified by ethanol precipitation. After purification, the samples were resolved on 5% polyacrylamide sequencing gels. The gels were...

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